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## Review

## Food and drug addictions: Similarities and differences



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## ABSTRACT

This review examines the merits of 'food addiction' as an explanation of excessive eating (i.e., eating in excess of what is required to maintain a healthy body weight). It describes various apparent similarities in appetites for foods and drugs. For example, conditioned environmental cues can arouse food and drug-seeking behaviour, 'craving' is an experience reported to precede eating and drug taking, 'bingeing' is associated with both eating and drug use, and conditioned and unconditioned tolerance occurs to food and drug ingestion. This is to be expected, as addictive drugs tap into the same processes and systems that evolved to motivate and control adaptive behaviours, including eating. The evidence, however, shows that drugs of abuse have more potent effects than foods, particularly in respect of their neuroadaptive effects that make them 'wanted.' While binge eating has been conceptualised as form of addictive behaviour, it is not a major cause of excessive eating, because binge eating has a far lower prevalence than obesity. Rather, it is proposed that obesity results from recurrent overconsumption of energy dense foods. Such foods are, relatedly, both attractive and (calorie for calorie) weakly satiating. Limiting their availability could partially decrease excessive eating and consequently decrease obesity. Arguably, persuading policy makers that these foods are addictive could support such action. However, blaming excessive eating on food addiction could be counterproductive, because it risks trivialising serious addictions, and because the attribution of excessive eating to food addiction implies an inability to control one's eating. Therefore, attributing everyday excessive eating to food addiction may neither explain nor significantly help reduce this problem.

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## 1. Introduction

The scientific use of the term addiction in reference to food (chocolate) has been traced back to 1890, followed by sporadic interest in the topic dating from the 1950s, and a burgeoning of publications in the area much more recently (Meule, 2015). This recent research comprises behavioural and physiological studies in humans, and the development of animal models of ‘food addiction’ which draw on extensive findings from animal models of drug addiction. A great part of the importance of addiction, of course, lies in the harm done to people with addictions, to their families and to others who are indirectly affected, plus the burden placed on healthcare providers and civil and government authorities. The individual and economic costs of overweight and obesity, with their associated conditions such as type 2 diabetes, cardiovascular disease and osteoarthritis, are also enormous, requiring ‘urgent global action’ (Ng et al., 2014). Linking these problems is the possibility that excessive eating (defined as food intake in excess of that required to maintain a healthy body weight) might be understood, at least in part, as food addiction. The purpose of this review is to assess the extent to which there are commonalities between the consumption of foods and consumption of addictive drugs such as alcohol, opioids, stimulants and tobacco, and whether this comparison could be helpful in combating excessive eating.

## 2. What is addiction?

This question is of course fundamental to deciding whether or not a particular behaviour, such as eating chocolate or smoking a cigarette, qualifies as an addiction. If, for example, very strict criteria were applied then perhaps it would be concluded that food addiction was rare or non-existent.

In medicine criteria for addiction are set out in, for example, the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5) (American Psychiatric Association, 2013) and the International Statistical Classification of Diseases and Related Health Problems (World Health Organization, 1992). These two manuals are largely in agreement in listing key criteria defining addiction as the presence of at least two or three of the following: difficulties in controlling substance use; a strong desire or craving for the substance; tolerance such that increased doses of the substance are required to achieve intoxication or the desired effects; adverse effects of acute withdrawal from the substance; neglect of alternative interests, and social, family and occupational activities; unsuccessful attempts to quit use; and continued use despite knowledge of physical or psychological harm caused by the substance. Actually, both manuals avoid using the term addiction, instead preferring ‘Substance Use Disorders’ and ‘substance use dependence,’ respectively. Others restrict addiction to ‘the extreme or psychopathological state where control over drug use is lost,’ and distinguish this from dependence which they say ‘refers to the state of needing a drug to function within normal limits’ and which ‘is often associated with tolerance and withdrawal, and with addiction’ (Altman et al., 1996, p 287).

Complementary to expert views, dictionary definitions provide very good evidence of how words are used in everyday life. The main dictionary definition of addiction can be summarised as ‘being physically and/or mentally dependent on a particular substance or activity,’ with dependence in this context defined as ‘being unable to do without something.’ Associated with these definitions are the concepts of ‘compulsion’ and ‘obsession,’ or more mildly a ‘fondness’ or ‘passion’ for something. The latter might apply to a hobbyist or, for example, someone who says they are ‘addicted to watching soap operas,’ communicating their affection for certain TV drama serials, but perhaps also hinting that they feel they spend proportionally too much of their time on this activity. Similarly, a person claiming to be a ‘chocoholic’ is probably ambivalent about what they perceive to be their excessive consumption of chocolate (Rogers and Smit, 2000).

However, there can be little doubt that these examples denote less serious difficulties resulting from ‘addiction’ than those faced by a person with a serious gambling problem or a person with Alcohol Use Disorder as defined in DSM-5.

This points to the necessity of considering the relative risk of addiction associated with exposure to different substances and activities, rather than categorising the substance as either addictive or non-addictive. For example, most consumers of alcohol do not become addicted, but some do. Although drinking coffee poses an even lower risk of addiction, a very small proportion of caffeine consumers probably do meet stringent criteria for substance dependence (addiction) (Strain et al., 1994). Note, however, that based on Altman et al.’s (1996) definition of dependence (above), a very large majority of the world’s caffeine consumers are dependent on caffeine (Rogers et al., 2013). In relation to foods, a key determinant of reward value appears to be energy density (calories per unit weight, Section 4.3), yet there is even a well-documented case of carrot addiction (Kaplan, 1996). So, depending on individual vulnerabilities and circumstances, a very large range of substances and activities must be considered as potentially addictive.

Above, addiction is defined primarily on the basis of behaviour towards substances and activities, together with reports of associated cognitions, emotions and other experiences. These behavioural tendencies and experiences will be represented in the brain but, more than that, drug use modifies brain chemistry in ways that perpetuate and potentially escalate consumption (Robinson and Berridge, 1993; Altman et al., 1996; American Psychiatric Association, 2013). In particular, drug-induced neural changes in cortical and basal ganglia structures, involving for example dopaminergic, GABAergic and opioid peptidergic neurocircuitry, are thought to be critical in the development of drug addiction (Everitt and Robbins, 2005; Koob and Volkow, 2016). These changes characterise the transition from occasional, voluntary drug use to habitual use, compulsion and chronic addiction and, together with heightened stress, underlie what is described as the three-stage recurring cycle of addiction, namely ‘binge/intoxication,’ ‘withdrawal/negative affect’ and ‘preoccupation/anticipation (craving)’ (Koob and Volkow, 2016). This is significant because much of the literature on food addiction considers food addiction to be similar to drug addiction

**Table 1**  
Some possible similarities in characteristics of appetites for foods and drugs.

Foods	Drugs	Section(s)
External cue control of desire to eat, including specific appetites	Cues associated with drug-taking increase desire for drug taking and acquire ‘incentive salience’	3.1, 3.8
Appetite comes with eating	Priming	3.2
Disinhibition of dietary restraint	Abstinence violation effect	3.3
Food craving	Drug craving	3.4
Tolerance to the physiologically disruptive effects of food ingestion, ‘satiety tolerance,’ etc.	Drug tolerance	3.5
Adverse effects of acute food withdrawal	Adverse effects of drug withdrawal	3.6
Bingeing on foods	Bingeing on drugs	3.7, 3.6, 4.1, 4.2
Liking and wanting for foods	Liking and wanting for drugs	3.8, 3.9, 4.3
Reward deficiency in obesity	Reward deficiency resulting from exposure to drugs	3.9

(e.g., Avena et al., 2008; Johnson and Kenny, 2010; Gearhardt et al., 2011a) rather than to behavioural addictions. The next question then, is to what extent do foods and drugs have common effects on behaviour and the brain?

### 3. Similarities and differences in appetites for foods and drugs

Table 1 summarises some possible similarities in characteristics of appetites for foods and appetites for drugs. These are framed as behavioural characteristics, however where applicable, evidence on underlying neurobiological mechanisms is also summarised. Listing does not imply close similarity, and where they exist, differences between foods and drugs in the characteristics are discussed.

#### 3.1. External cue control of appetites for foods and drugs

It is very well established that exposure to the sight and smell of food, and to arbitrary external stimuli previously associated with eating, increase desire to eat and appetitive behaviour (Rogers, 1999). The same cues also trigger physiological events, including increased salivation, gastric acid secretion and insulin release (Woods, 1991). It is possible that these responses feedback to, at least in part, cause the increase in appetite, although their main role would appear to prepare the body for food ingestion (Section 3.5). However, the effects, even of tasting food (Teff, 2011), are much smaller than the parallel physiological effects that follow ingestion. Exposure to food-related cues also acts as a reminder of eating and the pleasure of eating, and it appears that appetite is increased most for the cued food itself or a similar food, or food specific to that situation (e.g., in the UK often cereal or toast for breakfast, and popcorn in a cinema) (Rogers, 1999; Ferriday and Brunstrom, 2011).

Similarly, there is an extensive literature demonstrating the effects of drug-related cues on behaviour and physiology. The effects include increased craving for drugs in drug users exposed to drug-related stimuli, and reinstatement of responding for drugs in animals after a period on non-reinforced responding (extinction) and, more relevant to human drug use, after prolonged abstinence without extinction (Altman et al., 1996; Koob et al., 2014). As for food, these cues are reminders of drug use, and they can elicit conditioned drug-like and drug-opposite physiological responses (Altman et al., 1996). Also, with repeated drug use, drug users may become increasingly sensitised to the incentive properties of drug-associated cues (Robinson and Berridge, 1993; Section 3.8). Exposure, that is administration or self-administration, of a small amount of the drug itself can have even more powerful effects than drug-related cues. This is essentially priming, which is discussed next (Section 3.2). In the case of the oral consumption of a drug, alcohol, for example, the first mouthful or few mouthfuls combine exposure to flavour cues (arguably external cues) with a priming dose of the drug.

It can be expected that the effects of external cues will be modulated by the individual's current state of satiation (fullness in respect of eating and intoxication in respect of drug use). However, the observation that external eating-related cues can motivate consumption even in apparently sated rats and people (Weingarten, 1983; Cornell et al., 1989) should not be taken as evidence that external cues are 'overriding' internal regulatory signals (cf. Petrovich et al., 2002). This is because the spontaneous cessation of eating (which is the test of satiation) usually occurs before the gut is filled to capacity, so that at the end of a meal there is almost always likely to be 'room for more' if further food is presented (Rogers and Brunstrom, 2016). External food-related cues signal the opportunity to eat, and the capacity to store nutrients in excess of immediate needs allows such opportunities to be exploited, and it also allows meals to be missed without adverse effects. This contrasts with the more limited capacity to tolerate drug overdoses and drug withdrawal.

#### 3.2. The appetiser effect and priming

The phrase *l'appétit vient en mangeant* (appetite comes with eating) recognises the experience that the first mouthful of a liked food in a meal increases motivation to eat. This has been investigated by Yeomans (1996), who termed the phenomenon the 'appetiser effect.' Experiments with mice indicate a similar positive feedback effect of oral contact with food, the function of which may be to keep behaviour 'locked in' to eating, thus preventing its premature interruption by another activity (Wiepkema, 1971). As the meal progresses the positive feedback, which might involve both taste and early post-ingestive signals (de Araujo et al., 2008), is gradually outweighed by negative feedback arising from the accumulation of food in the gut (Rogers, 1999). Another example of eating-related priming (appetite 'whetting') is a study by Cornell et al. (1989). Behaviourally at least, the appetiser effect, although relatively small, is similar to what is referred to in the literature on drug addiction as priming effects, and the fact that this also occurs with food is noted in that literature (e.g., de Wit, 1996). In even a current long-term abstinent drug user, taking a small amount of the drug increases desire for the drug. In this context priming is of concern because it is liable to precipitate full relapse to drug use. This supports the tenet of complete abstinence recommended in many drug abuse treatment programmes.

#### 3.3. Disinhibited eating and the abstinence violation effect

Also involved in relapse are eating disinhibition and the related abstinence violation and snowball effects (Baumeister et al., 1994). These phenomena refer to unintended or greater than intended consumption, and are conceptualised primarily in terms of the cognitions and emotions involved in violation of abstinence goals. At the extreme, even minor transgressions are felt as catastrophic, which then undermines further efforts at self-control. This behaviour is exemplified by the following item on a widely applied eating disinhibition scale: 'While on a diet, if I eat a food that is not allowed, I often splurge and eat other high calorie food' (Stunkard and Messick, 1985). Behind this is an all-or-none style of thinking: 'What the hell, I've blown my diet, I might as well continue eating — I can always start (dieting) again tomorrow.' Both in relation to eating and drug use a recommendation is to direct attributions for goal violation (relapse) to controllable situational factors (e.g., one is expected to eat cake at a birthday party), rather than internal, stable factors such as lack of willpower, or addiction or disease (Baumeister et al., 1994). It is also the case that low mood and stress can trigger disinhibition and relapse, potentially in part by depleting cognitive resources. Mood- and stress-related eating are prominent items in the eating disinhibition scale. Eating disinhibition is a strong predictor of overweight and obesity (Bryant et al., 2008).

#### 3.4. Craving

Food and drug craving are defined as a strong desire or urge to consume a specific food or drug (Rogers and Smit, 2000; West and Brown, 2013), and as such craving denotes a subjective experience associated with eating and drug use. Measurement of craving therefore depends on spontaneous verbal self-reports of the experience, and answers on suitably-worded rating scales. This does not preclude the use of craving as a construct to describe behaviour in animals (e.g., it might be operationalised as rate of responding for drug reward), or indeed in humans, but its significance in relation to human motivation to consume foods and drugs lies in the extent to which craving represents a cause of appetitive behaviour and consumption, or a consequence of attempts to abstain from consumption. Certainly, drug use, for example smoking a cigarette, and eating can occur without being preceded by craving (Tiffany, 1995; Altman et al., 1996; Rogers and Smit, 2000). Indeed, eating is mostly not associated with craving. Instead, we might say that 'I'm hungry' when anticipating a meal, or that 'I was hungry'



when explaining why we ate a lot of food. Even this, though, is an exaggeration, as for adequately nourished people, readiness to eat is actually controlled by the absence of fullness (a full stomach inhibits appetite) rather than a short term deficit in energy supply to the body's organs and tissues (Rogers and Brunstrom, 2016).

Craving is, nevertheless, reported for certain foods, for example in the UK and the US most frequently for chocolate and other foods that are regarded as 'treats.' The attitude is that such foods should be eaten in limited quantities because, while delicious, they are also perceived as 'fattening,' 'unhealthy,' 'indulgent', etc. (i.e., 'nice but naughty'). Restricting intake causes elaboration of thoughts about the food and preoccupation with the prospect of eating it. These cognitions and associated emotions are then labelled as craving, or 'moreishness' (left desiring more) if the restriction occurs during an eating bout so as to curtail eating before inhibition of appetite by fullness (Rogers and Smit, 2000). This analysis is reminiscent of Tiffany's (1995) proposal that drug use is controlled largely by automatic processes and without the presence of the experience of craving unless drug use is prevented or resisted. Thus ambivalent attitudes towards certain foods and drug use and resulting attempts to restrict intake or fully abstain play a substantial role in causing both food and drug craving.

### 3.5. Tolerance

Drug tolerance is the reduction in the effect of a drug resulting from repeated exposure to the substance. Or operationally, it is 'a shift to the right in a dose-response effect function so that higher doses (of the drug) are required to produce the same effect' (Altman et al., 1996). Tolerance can occur to the rewarding as well as aversive effects of drugs of abuse, and it results from various adaptations, including to drug metabolism and target receptor function, and the development of conditioned (learned) anticipatory responses that oppose certain effects of the drug (Altman et al., 1996). Tolerance varies across drugs, and also varies for different effects of a drug, even to the extent that sensitisation (an increase in sensitivity) may occur to some effects (Altman et al., 1996). As an everyday example, the effects of caffeine demonstrate variation in tolerance. Complete or almost complete tolerance to the wakefulness and mild anxiogenic effects of caffeine occur at fairly modest levels of dietary exposure to caffeine (2–3 cups of coffee per day). By contrast there is only partial tolerance to the increase in hand tremor caused by caffeine, and little or no tolerance to the motor speeding (or endurance) effect of caffeine (Rogers et al., 2013). In general, tolerance to the adverse and aversive (side) effects of drugs, including tobacco, alcohol and opiates, is important in the initiation and maintenance of drug use and abuse (Altman et al., 1996). Tolerance to the rewarding effects of drugs may also increase consumption (Altman et al., 1996; West and Brown, 2013), but usually if a behaviour (i.e., drug or food ingestion) becomes less rewarding, over time, responding can be expected to decline (Rogers and Hardman, 2015). This is discussed further below in relation to 'reward deficiency' (Section 3.9).

In his review 'The Eating Paradox: How We Tolerate Food,' Woods (1991) makes an explicit link between drug and food tolerance. He argues that the so-called (conditioned) cephalic phase responses of salivation, gastric acid secretion and insulin release that occur in anticipation of eating serve to prepare the body for the physiological challenge of food ingestion. In doing so, they help maintain body homeostasis, akin to the function of conditioned drug tolerance. The identity of the responses differ between food and drug use and across drugs, and at least for food the magnitude of the anticipatory effects is smaller than the physiological responses to food in the mouth and after swallowing.

Another aspect of food tolerance is the increase in gastric capacity related to binge eating (Geliebter and Hashim, 2001). This might underlie 'satiety tolerance,' which would facilitate the consumption of larger meals over successive binges. Similarly, satiety tolerance might develop, although more gradually, in individuals who increase their meal size and/or meal frequency progressively over time, but who do so without

bingeing. In contrast, restricting intake will likely increase satiety sensitivity and in turn help perpetuate undereating in, for example, people with anorexia nervosa (restricting type). Illustrating this, salivation to food (but not to non-food odours) 2 h after eating breakfast was found to be increased in people with bulimia nervosa and decreased in people with anorexia nervosa, compared with controls. When eating patterns were, to a large extent normalised following 60 days of intensive in-patient treatment, these differences in salivation to food stimuli were greatly reduced (LeGoff et al., 1988). Lastly, tolerance to the inhibitory effects on appetite of increased body fat (e.g., 'leptin resistance') may be another contributing factor to excessive weight gain (Rogers and Brunstrom, 2016; Section 3.9).

Adaptation of both conditioned and unconditioned responses to the consumption of food and drugs functions to preserve body homeostasis. Relatedly, however, tolerance also contributes to the escalation of consumption and, at least in part, it similarly underlies the adverse and aversive effects of drug withdrawal (Altman et al., 1996). Both tolerance and withdrawal are criteria included in the definition of addiction. Withdrawal is described in the next section.

### 3.6. Withdrawal

An extended period of voluntary or forced abstinence from drug taking can result in adverse effects, including dysphoria, anxiety, insomnia, fatigue, nausea, muscle pain, autonomic effects and even seizures (American Psychiatric Association, 2013). The severity of withdrawal effects vary markedly across drug class, with withdrawal from alcohol and opioids having the worse effects. Escape from and avoidance of adverse withdrawal effects appear to play a significant role in maintaining drug use (Altman et al., 1996; Koob and Volkow, 2016) and, for example, nicotine replacement therapy which aims to reduce withdrawal effects associated with smoking, substantially increases success of quitting smoking (Stead et al., 2012). Also, using the example of caffeine once again, evidence points to caffeine consumption being very largely motivated by withdrawal reversal. This is in respect of both maintenance of wakefulness and cognitive performance (Rogers et al., 2013), and negatively reinforced liking for the taste of the vehicle (tea, coffee, etc.) in which the caffeine is consumed (Section 3.8).

Given that eating often occurs in the absence of immediate need for nourishment (which for most people in food-rich environments is most of the time), it cannot reasonably be equated with withdrawal relief. Nevertheless, in the absence of fullness, eating is rewarding (Rogers and Hardman, 2015), and therefore food abstinence or restriction means missing out on food reward, which is potentially both hard to resist and distressing.

An example of the effects of withdrawal of food reward is findings on rats offered intermittent access to 25% glucose or 10% sucrose solutions (cola and other soft drinks contain about 10% sucrose, and 'energy' drinks contain about 10% glucose) (Colantuoni et al., 2002; Avena et al., 2008). In these studies, rats given access to glucose and standard laboratory rat food (chow) for 12 h a day were compared with other groups of rats given, for example, continuous access to glucose and chow, or continuous access to only chow or intermittent access to only chow. When exposed to intermittent access the rats initially lost weight, but subsequently were able to increase their food intake to avoid further weight loss (Colantuoni et al., 2002). It is argued that the glucose-plus-chow-intermittent-access rats over time came to exhibit signs of addiction to sugar. Thus they are described as 'bingeing' on sugar, particularly when it became available at the beginning of the 12-hour period of access. For instance, glucose intake over the first 3 h of access increased from 8 ml on the first day of intermittent access to 30 ml on day 8. However, if this is the development of bingeing, the rats also binged on chow, because there was a parallel increase in chow intake (from 2.7 g on day 1 to 10.5 g on day 8) (Colantuoni et al., 2002). In any case, it is an exaggeration to call the first meal of sucrose consumed after daily deprivation a 'binge,' because this only

amounts to about 5% of total daily energy intake (Avena et al., 2008). Another way to describe this behaviour is that it represents adaptation to restricted access to food. With repeated experience of the intermittent access the rats are able to predict availability and this facilitates conditioned and unconditioned tolerance to larger meals of sugar and of chow (Section 3.5).

More compellingly, Avena et al. (2008) find similarities between the effects of drug withdrawal and the effects of withdrawal of access to sugar (plus chow). The model is the effect of withdrawal from opiates precipitated by administration of the opiate antagonist naloxone, which causes distress as indexed by, for example, behavioural depression and anxiety, measured respectively by the forced-swim test and time spent in the open arms of an elevated plus-maze. After naloxone, intermittent-sugar-and-chow-access rats (21 days access) showed worse 'withdrawal' on these measures than did the various control groups, although for the forced swim test the intermittent-chow-only group was intermediate between the intermittent-sugar-and-chow and ad libitum fed groups (Avena et al., 2008). Other studies in this series revealed further neuroadaptations in response to intermittent glucose and chow feeding having similarities to effects of exposure to drugs of abuse. These included changes indicating altered brain dopamine function, for example increased D1 and D2 receptor binding in the dorsal striatum, and increased D1 receptor binding in the core and shell of the nucleus accumbens (Avena et al., 2008). It was also found that dopamine release in response to drinking sugar remained elevated across 21 days of intermittent-sugar-plus-chow feeding, compared with a diminished dopamine response over time in the intermittent-chow group and other control groups (Avena et al., 2008) that is typical when an appetitive stimulus loses its novelty.

The authors' conclude that 'The evidence supports the hypothesis that under certain circumstances rats can become sugar dependent' (i.e., addicted, as indicated by the title of their paper) (Avena et al., 2008, p 20). This is plausible to the extent that intermittent access to, and withdrawal from, a rewarding food (sugar) under circumstances of repeated food deprivation, in an otherwise unstimulating environment, is highly significant. Further, this may model some of the features of binge eating after a period of (usually) self-imposed food restriction (Sections 3.5 and 3.7). Importantly, however, intermittent sugar plus chow access rats do not eat excessively and do not become overweight (Avena et al., 2008). By contrast, humans most at risk of excessive eating have continuous access to palatable food. In this context (unrestricted access), research on animals shows significant differences in neural responses to sugar and drugs. For example, dopamine release in the shell of the nucleus accumbens habituates rapidly in response to the consumption of sugar and other palatable foods, but not to addictive drugs, including morphine, alcohol and nicotine. Further, cues predictive of palatable foods and drugs similarly stimulate dopamine release in the medial pre-frontal cortex, but only cues predictive of drugs have this effect in the nucleus accumbens (Di Chiara, 2005). Other studies find differences in cell firing patterns in the nucleus accumbens of rats responding for cocaine versus food or water, which it is suggested may originate in neuroadaptation brought about by chronic drug exposure (Carelli, 2002).

While the relevance of intermittent access models to the human condition is questionable, it is the case that continuous access to a diet consisting of foods high in fat, and high in both fat and sugar, does lead to substantial increases in energy intake and body weight. This is discussed below in Section 3.9.

### 3.7. Bingeing

Binge eating is defined as 'eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances,' coupled with 'a sense of lack of control over eating during the episode.' (American Psychiatric Association, 2013). Binge

eating is characteristic of people with bulimia nervosa and binge eating disorder (BED), and it may also occur in people with anorexia nervosa. Binge drinking, referring to the rapid consumption alcohol to the point of inebriation, is perhaps a parallel example for drug use, although a difference is the effects of alcohol on decision making and attention (e.g., 'alcohol myopia') (Gable et al., 2016). More generally, any intoxication with a drug of abuse might equated to a binge (Koob et al., 2014).

For the present discussion, however, the significance of binge eating lies in it potentially fulfilling key criteria for addictive behaviour beyond excessive consumption, beginning with the sense of loss of control, but also including experiencing strong impulses to binge eat, pleasure or relief at the time of binge eating, tolerance (Section 3.5), and continued binge eating despite knowledge of persistent adverse effects. On this basis, in one study 92% of women diagnosed with BED fulfilled adapted DSM-IV criteria for substance dependence (addiction), although less than half that number (42%) met more stringent criteria for addiction (Cassin and von Ranson, 2007).

Nonetheless, food addiction as exemplified by binge eating would not appear to account for most of the excess eating that contributes to overweight and obesity. People with anorexia nervosa are, by definition, underweight, and while bulimia nervosa and BED are associated with overweight and obesity, their prevalence (e.g., respectively 1–1.5% and 1.6% of women in the US (American Psychiatric Association, 2013)) is much lower than the prevalence of obesity (e.g., currently about 37% in women in the US) within the same populations (cf. Epstein and Shaham, 2010; Ziauddeen et al., 2012).

### 3.8. Liking and wanting as motives for substance use

In their influential analysis of drug addiction, Robinson and Berridge (1993) distinguish between drug liking and wanting, and Berridge (1996) provides a parallel analysis for eating motivation (food reward). Drug liking is the 'subjective pleasurable effects' of the drug and is distinguished from the incentive motivational effects of drug-related stimuli, or wanting. Activation of nucleus accumbens-related neural circuitry underlies the attribution of 'incentive salience' to reward-relevant stimuli ('making them wanted'), and with repeated use of certain drugs this system becomes sensitised. By contrast, repeated use may cause drug liking to be diminished. The result of increased wanting is compulsive drug seeking and taking, despite the reduced pleasure in the effects achieved. It is plausible that similar neuroadaptations underlie excessive eating, perhaps in particular binge eating. In research on human eating behaviour, however, measurement of liking and wanting tend to be confounded. While it is reasonably straightforward to assess food liking by asking for a person's evaluation the pleasantness of the 'taste' of a food, so-called measures of wanting are probably really measures of 'food reward' (i.e., liking plus wanting) (Rogers and Hardman, 2015). Nonetheless, it does appear that liking and wanting largely affect food reward independently in that, for example, food reward but not food liking is increased by not having eaten for several hours. Distinct nucleus accumbens opioid 'hot spots' have been identified for liking and wanting (increased eating without increased liking) (Peciña and Berridge, 2005), and other more recent research has demonstrated elegantly how taste and nutrient components of food reward are also signalled by separate brain dopamine-signalling pathways (Tellez et al., 2016).

Food liking, though, would appear to differ somewhat from drug liking. Food liking is the pleasure (affective or hedonic response) generated primarily by oral contact with a food stimulus, whereas drug liking appears to refer to effects generated post-ingestively. For certain drugs, however, most notably, caffeine, alcohol and nicotine, administration combines both of these aspects of liking. For the coffee, beer, wine and whiskey drinker, and for the smoker of cigarettes and cigars, oro-sensory effects are important features of the pleasure of consumption, to the extent that there can be a high degree of discrimination between brands and varieties. The effects (sensations), including the

bitterness of caffeine and other compounds in coffee, the burning effect of alcohol in the mouth and the 'scratch' of nicotine on the throat, are initially aversive and disliked, but appear to acquire positive hedonic tone as a result of their consumption being paired with the respective drug's post-ingestive effects. This has been demonstrated for caffeine, which was found to reinforce liking for arbitrary flavours (fruit 'teas' and fruit juices) paired with caffeine intake (Yeomans et al., 1998), although this occurs only for caffeine consumers acutely deprived of caffeine, indicating negative reinforcement. In this way, drug-reinforced liking for the oro-sensory effects of a drug and its vehicle can come to act as an additional motive for consumption, as will inclusion of (congenitally liked) sweetness, via sugars or other sweeteners, in coffee, tea, etc. and in tobacco and alcohol products. Relative to wanting, however, the importance of this oro-sensory hedonic motive for consumption is much diminished in addiction (e.g., in Alcohol Use Disorder).

### 3.9. Reward deficiency

Reward deficiency (or deficit), or reward 'hyposensitivity,' refers to the idea that reduced drug and food reward causes compensatory overconsumption of these commodities (Blum et al., 1996; Wang et al., 2001; Johnson and Kenny, 2010; Stice and Yokum, 2016). (This is not the same as reward sensitivity in Gray's reinforcement sensitivity theory (Corr, 2008), although they may overlap). Individual differences in reward sensitivity potentially predict vulnerability to addiction, but more than this it is proposed that exposure to addictive drugs and certain foods causes neuroadaptations, primarily downregulation of striatal dopamine D2 function, that reduce reward sensitivity. In turn, this causes an escalation of consumption and, in the case of exposure to energy dense sweet and high-fat foods, results in obesity. In support of this Johnson and Kenny (2010) conclude the following from their studies of the neurochemical and behavioural effects of giving rats 'extended-access' (i.e., access 18–23 h per day for several weeks) to such foods: '*The development of obesity in extended-access rats was closely associated with a worsening deficit in brain reward function*' (p 636); and '*Reward deficits in overweight rats may reflect counteradaptive decreases in the baseline sensitivity of brain reward circuits to oppose their overstimulation by palatable food. Such diet-induced reward hypofunction may contribute to the development of obesity by increasing the motivation to consume high-reward 'obesogenic' diets to avoid or alleviate this state of negative reward*' (p 639).

One problem with this and other related proposals concerning reward deficiency as a cause of excessive eating and obesity is the notion that reduced reward leads to increased consumption. More logically, consumption might be expected to be *reduced* if it is experienced as less rewarding (Rogers and Hardman, 2015), and indeed evidence on food intake in rat dietary obesity points in that direction. Rats switched to an energy dense diet immediately greatly increase their energy intake and consequently gain body weight (mainly fat). Over weeks, however, energy intake falls and the rate of weight gain is slowed. This indicates a negative feedback effect of fatness on appetite (leptin signalling is likely involved here) (Rogers and Brunstrom, 2016). This is further supported by the observation that when the energy dense diet is withdrawn and the dietary-obese rats are returned to just the standard chow diet, they significantly under eat compared with control rats always maintained on chow, until that is the previously obese rats' weight falls to match that of the control rats (Rogers, 1985). These dynamics can be viewed in terms of a balance between stimulation of appetite by the reward value (plus reduced satiety effect per calorie) of energy dense foods and the inhibition of appetite proportional to body fat content (Rogers and Brunstrom, 2016). Based on this interpretation, Johnson and Kenny's (2010) conclusions, can be re-written thus: *The development of obesity in extended-access rats was closely associated with reduced brain reward function; and reduced reward in overweight rats may reflect adaptive decreases in the baseline sensitivity of brain reward circuits to oppose their stimulation by palatable food. Such*

*obesity-induced reward hypofunction may oppose the development of obesity by decreasing the motivation to eat.* A further result in favour of this reanalysis is that in Johnson and Kenny's (2010) studies the reward deficiency, as measured by increased current threshold for brain self-stimulation reward (electrodes implanted in lateral hypothalamus), persisted many days beyond withdrawal of the energy-dense foods, in contrast to the effects found in similar experiments for withdrawal of heroin, cocaine and nicotine (Epstein and Shaham, 2010). Rather than being a direct effect of acute food withdrawal, the persistence of reward deficiency in the dietary-obese rats is in line with the gradual reduction of weight in these animals (Rogers, 1985).

More generally, the evidence on reward deficiency as an explanation for excessive eating and obesity is very mixed. This includes evidence from neuroimaging studies (Ziauddeen et al., 2012; Stice and Yokum, 2016), and behavioural studies. An example of the latter is a study that used the tyrosine/phenylalanine depletion method to acutely reduce brain dopamine function in human participants, which contrary to reward deficiency found, if anything, that depletion decreased appetite and food intake (Hardman et al., 2012). Furthermore, prospective imaging studies have tended to find that lower responsivity to food reward predicts lower future weight gain. Based on this, and evidence from many other types of studies, Stice and Yokum (2016), conclude that 'existing data provide only minimal support for the reward deficit theory,' but that there is 'strong support for the incentive sensitization theory of obesity' (p 447). Similarly, the proposal that individual differences in susceptibility to drug addiction due to reward deficiency are related to variation in dopamine D2 receptor function (Blum et al., 1990; Blum et al., 1996) has subsequently been disputed. In support, there is evidence showing that, for example, decreased dopamine D2 receptor binding increases vulnerability to abuse cocaine, and that it is also an effect of exposure to cocaine, which in turn contributes to the maintenance of drug use (Nader and Czoty, 2005). On the other hand, the association of the dopamine D2 receptor gene Taq1A polymorphism and alcoholism, originally reported by Blum et al. (1990), has not been confirmed (Munafò et al., 2007). It also seems clear that there is no meaningful association between this polymorphism and human fatness (Hardman et al., 2014).

## 4. Discussion

The analysis above shows that there is substantial overlap in the behavioural processes and brain mechanisms involved in eating and those engaged by psychoactive drug use and abuse. Differences are also apparent, for example in the nature and details of tolerance and withdrawal effects, although of course in these respects there will also be differences across classes of drugs. As is often noted, foods and drugs differ because eating is necessary for survival and drug use is not (e.g., Epstein and Shaham, 2010; Ziauddeen et al., 2012), but then a healthy diet does not have to include highly-energy dense foods (Epstein and Shaham, 2010) – indeed one is likely to be healthier if such foods are largely avoided.

Of course, similarities between motivation to obtain and consume foods and addictive drugs can be expected, as these drugs tap into the same processes and systems that evolved to motivate and control adaptive behaviours, including eating (Ziauddeen et al., 2012; Salamone and Correa, 2013). The strong implication is that certain substances 'hijack' these control mechanisms leading to maladaptive behaviour and harm, because they have particularly potent rewarding and neuroadaptive effects. Put more succinctly, 'brain pathways that evolved to respond to natural rewards are also activated by addictive drugs' (Avena et al., 2008, p 20). However, the fact that food-related cues and eating activate these pathways is not in itself evidence for food addiction. In large part that classification comes down to what qualifies as addiction and the differing potency of different drugs and different foods to cause the defined effects.



#### 4.1. More than a matter of definition

An instrument that has been used widely in research on food addiction is the Yale Food Addiction Scale (YFAS; [Gearhardt et al., 2009](#)). It is a self-report scale (i.e., a not a diagnostic interview) consisting of 25 items related to different 'symptoms' of addiction, including difficulties in controlling substance use (e.g., 'I find that when I start eating certain foods, I end up eating much more than planned'), adverse effects of withdrawal (e.g., 'I have had withdrawal symptoms such as agitation, anxiety, or other physical symptoms when I cut down or stopped eating certain foods'), tolerance (e.g., 'Over time, I have found that I need to eat more and more to get the feeling I want, such as reduced negative emotions or increased pleasure'), and persistent desire to quit, implying unsuccessful attempts to quit (e.g., 'I have tried to cut down or stop eating certain kinds of foods'). The term 'certain foods' is explained to respondents at the beginning of the questionnaire as follows: 'People sometimes have difficulty controlling their intake of certain foods such as,' followed by a list of foods categorised as sweets, starches, salty snacks, fatty foods and sugary drinks. The criteria for 'substance dependence' (addiction) are a symptom count of  $\geq 3$  out of a maximum of 7, plus endorsement of one or both 'clinical significance' items (e.g., 'My behaviour with respect to food and eating causes significant distress'). A method is also provided for calculating a continuous score which yields a symptom count 'without diagnosis' (of substance dependence).

A concern with the YFAS is that it appears to be over-inclusive in assigning certain eating and eating-related behaviours as evidence of food addiction. For example, some of the foods listed (e.g., bread, pasta and rice) are staple foods worldwide, and while such foods may well feature in eating binges, the more everyday notion that it can be difficult to cut-down on eating these foods is remote from the 'extreme psychopathological state' that some researchers view as a hallmark of addiction ([Altman et al., 1996](#); [Section 2](#)). The finding that YFAS scores are high in people with BED (reviewed by [Long et al., 2015](#)) does not validate YFAS as a measure of food addiction, because many people not suffering with BED also meet the YFAS criteria for food addiction. Nor do findings of neural correlates of YFAS scores ([Gearhardt et al., 2011b](#)) establish YFAS as a measure of food addiction. YFAS scores correlated with brain activation evoked by anticipated receipt of food (chocolate milkshake). This included greater activation in the anterior cingulate cortex, medial orbitofrontal cortex, amygdala and dorsolateral prefrontal cortex. While these results resemble patterns of brain activation found for exposure to drug cues, these responses are not themselves diagnostic of addiction. Merely, they indicate, for example, greater attractiveness of and resistance to consuming chocolate milkshake in people with high YFAS scores.

Recently, Gearhardt and colleagues have published an updated version of YFAS. They developed YFAS 2.0 ([Gearhardt et al., 2016](#)) in part to be consistent with the definitions of substance related and addictive disorders in DSM-5. Food addiction is determined by the presence of clinically significant impairment plus symptom count scores (maximum = 11) of 2 or 3, 4 or 5, and  $\geq 6$  representing mild, moderate and severe food addiction, respectively. Symptom count was found to correlate positively with body mass index and, for example, with scores on scales measuring binge eating and disinhibited eating. In most respects YFAS and YFAS 2.0 are quite similar, though prevalence for some symptoms is lower in YFAS 2.0 (e.g., 'cutting down' on consumption of certain foods), seemingly due to rephrasing of the contributing items.

Of course, despite the various objections voiced above, it could be argued that the YFAS (and YFAS 2.0) is a legitimate way to operationalise food addiction. However, at least a major part of the usefulness of addiction as a concept lies in the extent to which it can both explain excessive behaviour and guide interventions to successfully treat and avoid the problem (cf. [Long et al., 2015](#)). That may, or may not ([Fairburn, 2013](#)), hold true for treating BED as food addiction, or perhaps as 'eating

addiction,' as no single food is implicated ([Hebebrand et al., 2014](#)). By contrast, it may not be helpful to view obesity, in the absence of a diagnosis of BED, as a consequence of food addiction. The reasons for this are discussed next.

#### 4.2. Is food addiction a helpful or unhelpful explanation of obesity?

As previously described ([Section 3.7](#)), the prevalence of obesity is much greater than the prevalence of binge eating, so the greatest harm done by excessive eating is the effects of obesity on physical and psychological well-being. But food addiction does not appear to be a major cause of excessive eating responsible for obesity. For example, one study found that only 7.7% of overweight or obese participants met the, arguably lenient, YFAS criteria for food addiction, compared with 1.6% of under-weight and healthy-weight participants. In this sample of 652 people living in Canada the prevalence of overweight and obesity was 62% ([Pedram et al., 2013](#)). Clearly, energy intake in excess of energy requirements occurs more frequently in the absence than in the presence of food addiction.

This does not necessarily mean that insights from addiction research might not inform treatments for obesity, but equally it is possible that attributing obesity to food addiction may be counterproductive to the goal of eating less. Indeed, in his book *The Myth of Addiction*, [Davies \(1992\)](#) argues that the concept of addiction can be unhelpful even as applied to psychoactive drug use. For example he suggests a cycle in which exaggeration of the adverse effects of drug withdrawal serves to explain (excuse) continuing drug use. In turn, this escalates expectations about the severity of withdrawal, and so on. Similarly, the problem with believing that food restriction will cause one to feel impossibly hungry, to 'run out of energy,' or to feel irritable or agitated, is that this may well make dieting to lose weight more difficult than might otherwise be the case ([Rogers and Brunstrom, 2016](#)). Believing that one's impulse to eat, for example, ice cream or cake, is due to food addiction, implies that the impulse is uncontrollable, making it less likely that the ice cream or cake can be resisted (and cf. [Section 3.3](#)). Another example is that a shared belief in chocolate craving and the attribution of this to 'chocolism' may reduce one's motivation and capacity to eat less chocolate ([Rogers and Smit, 2000](#)). An illustration of the powerful influence of belief on the experience of appetite is a study in which participants were led to understand that a liquid food would gel in the stomach. This belief alone (without the gelling effect) increased perceived fullness, reduced subsequent eating, and it also affected release of gastro-intestinal hormones and reduced gastric emptying rate ([Cassady et al., 2012](#)).

This raises a question about the effect of labelling certain foods as addictive. In a recent study ([Hardman et al., 2015](#)) participants studied three passages in preparation for a later test of memory of their contents. The third passage was about food addiction, with half of the participants receiving a version claiming that food addiction was real and half receiving a version claiming it to be a myth. In what participants were led to believe was a separate study, they subsequently took part in a 'taste test' in which they evaluated four foods, and were then left alone for 10 min to eat as much of the foods as they desired. Intake of crisps and cookies (foods of the type that were implied to be addictive) was 31% higher (not significant) and significantly more variable in the addiction-is-real group than in the myth group. There were no differences in intake of the other two foods (grapes and breadsticks). A further result was that the manipulation affected self-diagnosis of food addiction – more participants in the addiction-is-real group answered yes to the question 'Do you perceive yourself to be a food addict?' than did participants in the myth group. One conclusion from this study is that external endorsement of the concept of food addiction encourages people to view themselves as food addicts, with the possible consequence that they will then be more likely attribute their eating to food addiction. The greater variability in intake of potentially



'addictive foods' points to two divergent effects of belief in food addiction, namely avoidance of the food for fear of losing control versus giving in to inevitable failure of control. Thus perceiving consummatory behaviours in terms of addictions can be helpful or unhelpful for avoidance of harm. Notably, it can be expected that the effect will depend on the stage of substance use. For example, for the young person contemplating taking up smoking tobacco, the idea that tobacco is highly addictive may prevent them from starting to smoke. However, for the 20-a-day smoker this knowledge is likely to deter attempts to quit.

#### 4.3. Addiction risk

As described earlier (Section 2), likelihood of addiction varies greatly across different substances. Heroin can be highly addictive, chocolate much less so. Notably, comparisons between effects of cocaine and food rewards found that food restricted rats chose food over intravenous infusion of cocaine on 70–80% of trials (Tunstall and Kearns, 2014). Cocaine and food delivery were paired with a different auditory cue. The cocaine-paired cue was found to re-instate responding after extinction more powerfully than did the food-paired cue. This result can be interpreted as indicating greater liking for food but greater wanting for cocaine (Tunstall and Kearns, 2014), consistent with cocaine presenting a higher risk of addiction than food. In respect of differences between foods it has been proposed that addiction is particularly associated with highly processed foods (Schulte et al., 2015). These are foods that tend to have a high glycaemic load (i.e., they are high in sugar and/or other refined carbohydrates), or are high in fat, or both. Arguably, the high attractiveness, or 'hyper-palatability' of such foods to a large extent lies in their taste characteristics, specifically their sweetness, saltiness and/or savouriness (umami taste), all of which are innately liked by humans, together with their high energy density. It has been proposed that energy dense foods acquire high reward value due to their high nutrient (primarily carbohydrate and fat) content to satiety ratio (Rogers and Brunstrom, 2016). This is because nutrient ingestion is the ultimate goal of eating, but satiety limits further intake. So high availability of energy dense foods is liable to promote excessive energy intake for two related reasons: they are attractive and they are weakly satiating calorie for calorie. However, this overconsumption of energy and consequent overweight and obesity mostly occur in the absence of addiction to these foods unless, that is, food addiction is loosely defined (Section 4.2).

Risk of addiction also varies across individuals (as does risk of obesity), and individual variation in reward responsivity was discussed in Section 3.9. Further analysis of individual differences in vulnerability to addiction is outside the scope of this review, except to note that many interacting factors are involved in determining an individual's risk of addiction (Altman et al., 1996; West and Brown, 2013). These comprise, for example, genetic, developmental, temperamental, environmental, socio-economic and cultural factors, and legal context. Included here is equality of access to non-drug (and non-food) rewards. Some of these risk factors are more readily modifiable than others.

In relation to excessive eating, environments in developed nations are saturated with food. The ubiquity of food cues and the almost effortless access to food, particularly to energy dense food, encourages consumption beyond immediate needs (Rogers and Brunstrom, 2016). Individual differences in motivation and capacity to resist food reward will, to an extent determine, who gets fat, but changes to food environments would do much to help those vulnerable to excessive eating. In the UK, for example, discounted energy dense food is actively marketed ('pushed') at checkouts, including in primarily non-food retail outlets. Perhaps eventually this practice will cease because, like for alcoholic drinks or tobacco products, doing this will be regarded as unacceptably harmful to public health.

#### 5. Final comments and conclusions

The present analysis indicates similarities, but also some differences, in the motivational effects of food and drugs of abuse. In general, addictive drugs have more potent effects than foods, particularly in respect of their effects on the brain that make them 'wanted.' Whilst arguably binge eating can be conceptualised as a form of addictive behaviour, binge eating is not a major cause of excessive eating, because it has a much lower prevalence than either overweight or obesity. Rather than being seen in terms of food addiction, excessive eating is better explained by the wide availability, attractiveness and lower satiating capacity (calorie for calorie) of energy dense foods. It has been argued that establishing the addictiveness of such foods would help to persuade policy makers and others to restrict the marketing and availability of such foods, as has been done successfully, for example, for tobacco with the consequent reduction in prevalence of smoking and smoking-related ill health (Gearhardt et al., 2011a). However, the broadening of the definition of addiction that this would require might substantially lessen its impact. Extending addiction to food in this way also risks trivialising serious addictions, or it might make certain foods (i.e., 'addictive foods') seem even more difficult to resist. It could even have all of these unintended effects.

Another illustration of how words matter is provided by the demonstration that the same volatile stimulus (1:1 mixture of isovaleric and butyric acids) is perceived as very much more pleasant if it is labelled as Parmesan cheese than if it is labelled as vomit (Herz and von Clef, 2001). Likewise, using 'craving,' to describe having a strong desire to eat chocolate, 'bingeing' to describe consuming a large (or not so large) meal, and being a 'food addict' to describe being prone to excessive eating, prompts different perceptions of these rather ordinary experiences. The concern is that conceptualising excessive eating as food addiction neither explains excessive eating nor offers strategies for successfully reducing excessive eating.

*'We must learn to handle words effectively; but at the same time we must preserve and, if necessary, intensify our ability to look at the world directly and not through the half-opaque medium of concepts, which distorts every given fact into the all too familiar likeness of some generic label or explanatory abstraction.'*

From The Doors of Perception, by Aldous Huxley.

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